

ciated with fever, malaise, dizziness, headaches, anemia and even death. WILLIAM ABRAMOVITS, MD

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Current Antifungal Therapy

CUTANEOUS FUNGAL INFECTIONS are frequently seen in an office based practice. They are mainly treated by topical agents.

Tinea versicolor, caused by *Pityrosporum orbiculare* (*Malassezia furfur*), is often treated with 2½ percent selenium sulfide suspension applied for ten minutes a day or overnight initially and less frequently subsequently. Acrisorcin (Akrinol) sodium thiosulfate is also effective.

Superficial mycoses caused most frequently by *Trichophyton rubrum*, *Trichophyton mentagrophytes* or *Epidermophyton floccosum* include tinea pedis, cruris or corporis. Several topical agents may be used successfully. Tolnaftate (Aftate, Tinactin) is a colorless, odorless compound available as a cream, liquid or powder. It has a cure rate of 73 percent to 93 percent and is available over the counter. Haloprogin (Halotex) is available as a 1 percent solution or cream, and applied daily gives 68 percent to 92 percent cure rates. Clotrimazole (Lotrimin) is an imidazole with a wide range of activity against dermatophytes, yeasts, filamentous and dimorphic fungi. Available as a 1 percent solution or cream, it gives a 59 percent to 85 percent cure rate. Miconazole (MicaTin) is also an imidazole and is effective against dermatophytes, yeast and Gram-positive bacteria and gives a 75 percent to 100 percent cure rate. It also has been found effective in the treatment of aspergillosis, coccidioidomycosis and cryptococcal meningitis. Haloprogin, clotrimazole and miconazole are available on prescription only.

Underlying factors which predispose to dermatophyte infection should be corrected. They include excessive perspiration and retention of sweat by tight fitting garments and shoes; obesity, and diabetes mellitus.

Oral administration of griseofulvin should be reserved for patients unresponsive to topical therapy. In its new form, the active ingredient is dispersed as ultramicrosized particles in a polyethylene glycol vehicle (Gris-PEG, Fulvicin-UIF). Since it does not require fat for absorption, it can be taken without meals. The enhanced absorption of the compound usually requires a dose of only 125 mg twice a day. Higher doses may be needed for recalcitrant and refractory infections.

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Future of Antiandrogens in Acne

IT IS WELL RECOGNIZED that the responsiveness of the skin to androgens is associated with many common disorders such as hirsutism, male pattern baldness and acne vulgaris. Although alterations in the keratinization and inflammatory reaction in the follicular duct play a role in the pathogenesis of acne, there is fairly good evidence that lipogenesis in the sebaceous gland is necessary for the disease to occur. The primary events in sebaceous gland lipogenesis appear to be controlled by androgens.

Thus, theoretically it would be reasonable to treat acne with topical agents that are antiandrogen in nature. Cyproterone acetate (CPA) has been used in Europe. Its mechanism of action is competition for the androgen receptor on the sebaceous gland cell. While Cunliffe and Pye have reported no success in its topical use, Winkler has reported decreased sebum secretion of sequential use. CPA with ethynol estradiol given orally results in distinct improvement of acne, but no reports on topical use are available. Flutamide is a similar compound which is nonsteroidal and is effective in animal studies, but has not been tried in humans.

Progesterone competitively blocks the conversion of testosterone to dihydrotestosterone, when applied to male pubic skin reduces 5-reductase activity, but does not reduce sebum output. Estrogen in large nonphysiologic doses reduces seba-

ceous gland size and sebum output, but their mechanism of inhibiting androgen action is unclear. Ethinyl estradiol applied topically reduces sebum production.

There are several controversies and some confusion in the literature regarding the activity of topical antiandrogens. These inconsistencies arise from the fact that different vehicles produce different percutaneous absorption, while some substances initially dissolve the antiandrogen, but later crystalize it, or are absorbed too rapidly. In addition, skin bacteria, proteins, lipids and other agents transform the antiandrogens before they

reach their site of action, namely the sebaceous gland cells.

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